early in the attack, 4000 to 6000 units being enough to produce the characteristic effect on the membrane. Dr. Osler[1] suggests that those in charge of institutions who still have any lingering doubts (concerning the efficacy of diphtheria antitoxin) should, in the interests of their little patients, and in a spirit of humility, visit the South Department of the Boston City Hospital, and learn a few salutary lessons from its director, Dr. McCollom.

REPORT OF A CASE OF FIBRINOUS BRONCHITIS, WITH A REVIEW OF ALL CASES IN THE LITERATURE.[2]

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Cases of fibrinous bronchitis, or plastic bronchitis, or bronchitis pseudomembranosa, or bronchial croup, are as interesting as they are rare. The following is the history of a case observed in the Maternity Ward of the Johns Hopkins Hospital in September last.

Maggie S., colored, aged twenty-two years; II.-para. Rather emaciated looking; very dull mentally.

Family History. No tuberculosis; father suffers from rheumatism; otherwise negative.

Past History. Labors normal. Menstrual history normal. Six years ago, in the fall, she had an attack similar to the attack observed in the hospital, the symptoms being cough, pain, respiratory distress, profuse expectoration, containing, evidently from patient's description, branching casts. Following this attack she had "pneumonia," being confined to bed one month. Every year in the fall since that time she has had similar attacks. These attacks had no relationship to her previous pregnancies, which occurred two and three years previous to entrance to hospital, respectively. (See Note I.) She has lost in weight throughout the last two years. She entered the hospital in the ninth lunar month of her pregnancy, on July 27th. At the time of her entrance she had a temperature of 101.5°, which, in two or three days, had dropped to normal. From this time until the time of her delivery there is no note on her history of any abnormal condition, and for the last twelve days of this period she was under personal observation and nothing abnormal was noted. She was delivered on September 12th. She came to the delivery-room with a normal temperature, no cough, respiratory distress, or expectoration.

Present Illness. Her puerperium was normal except for a slight cough and a slight evening temperature—100° to 101°. She had the signs of a light bronchitis, lengthened expiration, and crepitant râles at the bases in the back.

On the tenth day she was allowed up in her chair, but the discomfort from her cough increased so much that she was put to bed again, her evening temperature having touched 102°.

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[1] The Principles and Practice of Medicine, 1901, fourth edition, p. 155.

[2] Parts of this paper were read at the Johns Hopkins Medical Society on June 2, 1901, and specimens demonstrated.

On the thirteenth day of her puerperium I was in the ward delivering another case, and my attention was called by the nurse to the patient's extreme dyspnœa.

Physical Examination (during first attack, observed on September 25th). Patient is sitting up in bed in intense dyspnœa. All the accessory muscles of respiration are called into play. The alæ nasæ dilate with each respiratory movement. She coughs incessantly, and brings up large masses of expectoration. She is complaining of an intense sharp pain radiating from the lower left side through the back, increased on coughing. Respirations are 35 per minute; pulse, 100; temperature, 102°. Loose tracheal râles are heard in the throat. On palpation there is normal vocal fremitus throughout. On percussion there is high tympany in both lower axillary regions, higher and approaching dull on the right. On auscultation over the fronts respiration is prolonged and accompanied by fine moist râles. The breathing is more intense in the right lower axilla, and approaches tubular breathing in its quality. The inspirations are here accompanied by râles distinctly crepitant in character. There is impaired resonance in the right lower back and harsher and louder breath sounds, and in one region covered by the bell of the stethoscope the breathing is distinctly tubular and accompanied by crepitant râles. The attack of intense dyspnœa lasted about one hour. The expectoration contained the large casts, from which I have prepared sections, and some smaller incomplete casts. Three days after the first attack Dr. Futcher made a careful physical examination, with the following result:

Right Lung. Percussion note and breath sounds are particularly clear throughout the entire front. The note is slightly impaired over the lower subscapular and axillary region. Over the lower interscapular and throughout the subscapular region the breath sounds are enfeebled, and there are a few moist râles on expiration. The voice sounds are slightly impaired.

Left Lung. On percussion the note is clear throughout the upper front and distinctly impaired throughout the lower half of the interscapular region. On auscultation a definite pleuritic friction rub is audible over the second, third, and fourth interspaces in the parasternal line, and is audible out to the anterior maxillary line. The breath sounds are not tubular. In the third interspace there is a cardiac respiratory rhythm to the friction rub. Between the mid-axillary and anterior scapular lines, and extending from the apex of the axilla to the costal margin, there is a definite area of impaired resonance, over which the breath sounds are almost inaudible and the voice sounds enfeebled. In the lower intrascapular region there are fairly numerous medium moist râles, some of which approach a crepitant quality.

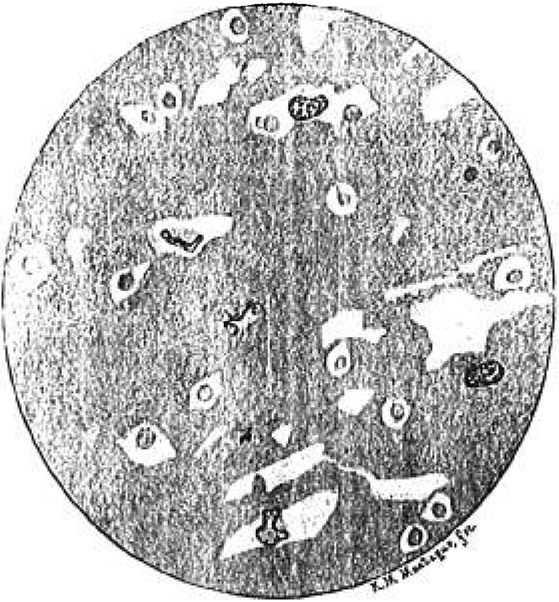
Heart. Point of maximum impulse is best seen and felt in the fifth interspace 10 cm. from the median line. There is normal cardiac dulness. Both sounds are audible at the apex. The first is accompanied by a very soft systolic bruit. In the third interspace a pleuropericardial friction rub is audible as noted above.

Leucocytes at this date were 5000, and the differential count was normal.

Further course of present illness: She had another paroxysmal attack, not so severe in character, on October 6th (eleven days later), in which she coughed up the large cast which is represented in the drawing, and several smaller less complete casts. (See Fig. 1.) The sputum expectorated during this attack on October 6th showed the following characteristics: Amount, 100 c.c.; separates into three layers on standing: (1) a granular detritus; (2) milky fluid; (3) branching cast.

First Layer. Cells; alveolar epithelium, leucocytes which were not eosinophiles.

FIG. 1.

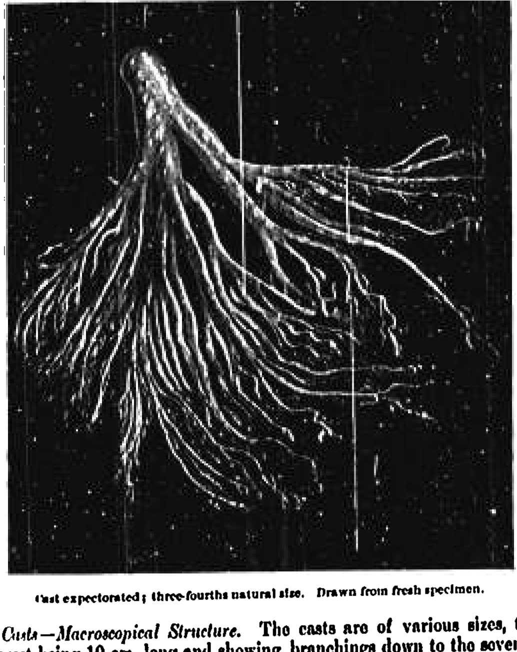


Second Layer. Bacteria, streptococci and diplococci; no tubercle bacilli, no Charcot-Leyden crystals, or Curschmann's spirals.

Third Layer. Branching casts which contained cells, the nature of which was not determined. No tubercle bacilli, no Charcot-Leyden crystals.

In the intervals between the attacks there was some slight respiratory distress and an evening temperature ranging between 100° and 101°. Only on a few occasions did the temperature reach 101°, so that a uterine culture was not taken, but puerperal infection could be definitely excluded by the absence of local signs. She had a slight cough, and occasionally expectorated small casts, which caused her very little discomfort. On one occasion a to-and-fro vibrating sound was heard on expiration in the right axilla. This corresponds in character to the "ventilgeräusch" of the Germans. The urine was negative throughout. The patient continued to have a slight cough and dyspnœa and slight evening temperature, and left the hospital in spite of our protests, October 10th. She remained in Baltimore for three weeks after her discharge from the hospital, and had frequent attacks of violent coughing and expectorated "tree-like things" (description of a friend). She then went to her home in Virginia, where she died two weeks later. No data as to the cause of death are obtainable.

FIG. 2.



Cast expectorated; three-fourths natural size. Drawn from fresh specimen.

Casts—Macroscopical Structure. The casts are of various sizes, the largest being 10 cm. long and showing branchings down to the seventh or eighth degree. (See Fig. 2.) They number about twelve. Their color is white, their consistency that of fibrin. Many show little intumescentia over the surface of the larger branches due to little bubbles of contained air. The morphology of the casts is best seen by looking at the illustration showing a portion of the cast embedded, from which the sections have been cut. (See Fig. 2.) It shows an outer stratified layer or skin, which encloses, in what would otherwise be a very large lumen, various separate whirls and cylinders. One immediately gets the impression from looking at this cross section that the exudation starts in the finer bronchi, and is then gradually pushed up into the larger bronchi, where additional exudation is apposed. That is, supposing the largest branch of a large cast to represent a cast of the bronchus of the third degree, the substance forming that cast represents not only exudation formed in that bronchus itself, but also that formed in all the bronchi below the level of this bronchus of the third degree. Various portions of the cast were hardened in Müller's fluid or by the formalin alcohol method, and were stained by the following methods:

1. Weigert's fibrin stain (celloidin sections). 2. Hæmatoxylin and eosin (celloidin sections). 3. Eosin and methylene blue (paraffin sections) (see Wright and Mallory). 4. Ziel-Nielson's method for tubercle bacilli (paraffin sections). 5. Unna's method for mucin (paraffin sections).

Results. 1. A surprisingly small portion of the sections take Weigert's fibrin stain. The greater number of fibrin fibrils are distributed in the outer layer or "skin" of the cast, or in the outer layer of the smaller whirls. The stain brings out organisms, streptococci and staphylococci, generally adhering to the outer side of the cast, though a very few are occasionally found in the mucus in the inside of the cast.

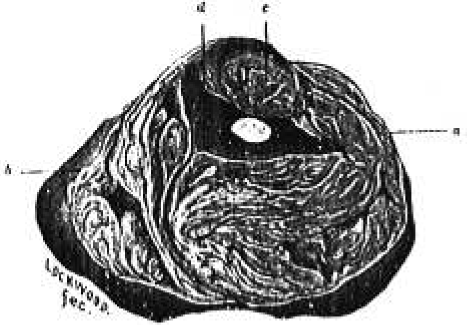
2. *Hæmatoxylin and Eosin.* This stain shows a ground substance consisting of two apparently distinct materials, a fibrillar substance taking a deep eosin stain, which is concentrically arranged about the lumina of the separate whirls. This encloses a second substance which is transparent and takes a light tinge of hæmatoxylin. Undoubtedly this latter substance, from its optical properties and from its staining reaction, is mucin. The substance taking the eosin stain so deeply is not altogether fibrin, but in part shows itself to be such, for it contains the fibrillæ which take the fibrin stain. What the nature of the portions that do not take the fibrin stain is it is difficult to say from the data at hand. Cells are very numerous, and are for the most part mononuclear leucocytes, though some polynuclears are seen. They are more numerous in the mucin ground substance than in the eosinophilic material. There are a few alveolar cells, many of which contain blood pigment; a very occasional red blood-cell is seen. Beside these cellular elements there are certain bodies which we supposed at first were cellular degenerations, but which in other stains took on a more specific character, and are, perhaps, some peculiar form of organism. These will be spoken of under 4. There were also a small number of cells showing eosinophilic granules. None of these are polymorphonuclear leucocytes, but are either degenerating epithelial cells or large mononuclear leucocytes.

3. *Sections stained in eosin and methylene blue.* These show the ground substance to be reticulated and taking altogether a deep eosin stain. The methylene blue stains only the nuclei of the cells. There are no mastzellen. This stain brings out well the few eosinophilic cells.

4. *The tubercle stain* showed no tubercle bacilli, but brought out those peculiar bodies which are mentioned above. They are irregularly round, from 7 to 15μ in diameter. (See Fig. 3.) They retain the fuchsin, keeping a deep red color. They have apparently a double

contoured outer shell, from which in certain places the deeply staining protoplasm seems to have shrunk away. Vacuoles of various sizes are seen in their protoplasm. The protoplasm stains diffusely, but shows a few more deeply staining granules. The shell seems to have a tendency to roll up on itself. These bodies were first seen in the sections stained for tubercle just four days before the reading of this paper at the Johns Hopkins Medical Society on June 2d, so that it is too early to give accurate descriptions of them or to judge as to their nature. The specificity of their staining and morphology suggests that they are some form of organism. After finding them here we looked for them in Weigert sections, and found that they retained Weigert's stain, showing the same characteristics with this stain as with the tubercle stain. They also take a specific stain in the eosin methylene blue method. In this they show well the double contoured, highly refractive outer shell, but none of the details of the protoplasm are brought out. In the hæmatoxylin and eosin stain these bodies look like degenerated nuclei, retaining the hæmatoxylin.

FIG. 8.



Drawn'under a magnification of 16, showing the portion of the cast at the level of its primary division into three branchings From the specimen embedded in celloidin, thus giving an idea of the cross section and the third dimension.

a and *b*. Outer skin. *c*. Separate whirl representing exudate from a smaller branch. *d*. Largest branching.

5. Unna's method with polychromethylene blue for mucin shows a substance taking a blue stain and a more opaque substance taking a beautiful red.

Cultures were made from the inside of the main branch of the largest cast after searing its surface, and streptococcus pyogenes and staphylococcus aureus obtained in pure culture. No diphtheria bacilli or pneumococci were cultivated.

Collections of all the cases in the literature have been made by Valleix (1843), Peacock (1854), Lebert (1869), Biermer (1867), Riegel (1875), West (1889). Lebert's article is very complete. It is entitled

"Ueber das Vorkommen fibrinoser entzündungsproducte in den Bronchien und Lungen-alveolen—Ueber fibrinose oder Pseudomembranose Bronchitis und Pneumonie, Bronchitis Fibrinosa, Bronchitis Pseudomembranacea, Pneumonia fibrinosa."

He excludes from consideration all cases secondary to diphtheritic inflammation of the pharynx and larynx. These are cases caused, as we now know, by the Klebs-Löffler bacillus. They are especially discussed by Peter, Millard, and Trousseau. Lepine, who reviews this whole subject, gives only one authentic case, which he cites from Trousseau. One is led to expect that actual cases are rare, for the subject receives always at the hands of French writers general consideration. It is most often seen following tracheotomy. Lebert comes back to the term "fibrinous bronchitis" as opposed to Biermer's "pseudomembranous bronchitis." His group of cases of imperfect observations—thirty-two in all—are interesting mainly from an historical stand-point. Galen evidently observed a case and considered the expectorated masses vessels of the lung. Donatus (1586) describes the casts as "venæ *frustum* parvum per tussum excreatum e pulmonone." Nicol Tulpius as "surculi venæ arteriosæ expectorati." Bussiere (1750) made an autopsy on a case in all probability associated with tuberculosis, and found a cast *in situ*, thus proving that they were not vessels of the lung, but bronchial secretion. In the early part of the eighteenth century the casts were commonly called bronchial polypi: thus Samber (1727) and Ruyschius (1721). The most interesting case in this group and unique in the whole history of the disease is that of Hayn (1844), who describes finding at autopsy in an infant, aged three weeks, extensive casts throughout the bronchi of both lungs. Lebert's second group of cases—five in all—are not cases of fibrinous bronchitis. They are fatal cases of bronchopneumonia in which at autopsy bits of membrane are found in the bronchi, never forming any definite branching casts, but being scattered here and there throughout the pneumonic areas. Lebert's third group are cases of acute fibrinous bronchitis including seventeen cases. He gives a careful analysis of these cases, four of which were fatal, and complete histories of seven cases, including the four fatal ones. Three of these four were fatal in an attack, and one died in typhoidal collapse. The cases of Lebert's fourth group, associated with pneumonia, are, indeed, remarkable, and very suggestive. He calls attention to the fact well established through the researches of Lobstein, Remak, and Rokitansky that in lobar pneumonia little fibrinous plugs are expectorated in a certain proportion of cases (40 per cent. according to Remak), and are frequently found at autopsy in the smaller bronchi. He then reports five cases out of the literature in which definite branching casts of the bronchi were found in pneumonia. Four of these five cases were fatal, and in three out of these four casts were never expectorated but were found at autopsy. All were fatal early in the disease. His fifth group of cases, including twenty-seven, are cases of idiopathic chronic fibrinous bronchitis. He gives a careful analysis of these, which it will be unnecessary to repeat, as the analysis of the cases given below covers the ground. There are seven cases in his sixth group of chronic symptomatic fibrinous bronchitis. Four of these are associated with the last stages of pulmonary tuberculosis; one with fatal abscess of the lung and mitral disease; one with syphilitic ulceration and stricture of the larynx, and one with empyema, this last case being the only one that was not followed to a fatal termination and autopsied.

I have tabulated all the cases that have been reported since the publication of Lebert's paper in 1869 in French, German, English, and American journals and monographs. Allow me to present an analysis of these cases. The cases reviewed fall naturally into the nine groups given below:

I. Chronic bronchitis with expectoration of branching casts of the bronchial tree: twenty-seven cases.

II. Acute bronchitis with expectoration of branching casts of the bronchial tree: fifteen cases.

III. Cases in which branching casts were not expectorated, but were found in the bronchi at autopsy: six cases.

IV. Cases in which the casts expectorated showed no dichotomous branching: eleven cases.

V. Expectoration of branching casts in organic heart disease: ten cases.

VI. Expectoration of branching casts in pulmonary tuberculosis: fourteen cases.

VII. Expectoration of small casts, often non-branching, in association with asthma: five cases.

VIII. Formation of casts in the bronchi in association with pulmonary œdema following thoracentesis: four cases.

IX. Cases whose classification is doubtful because of incomplete reports: six cases.

ANALYSIS OF CASES IN GROUP I. *Etiology.* Sex: Males, 15; females, 11; (?) 1.

Age: First decade, 1; second decade, 3; third decade, 5; fourth decade, 9; fifth decade, 4; sixth decade, 2; seventh decade, 2; (?) 1. This shows progressive increase up to middle life, and thence a decline.

Occupation: There are five cases in which occupation is given as possibly having some bearing: Street, "captain of the hold" in a ship; Stirling, dock laborer; Schittenhelm, grave-digger; Roque, blacksmith; Hall, painter.

Infectious Diseases. There is a history of previous pneumonia in Worthington's case, and also in Singer's, Sax's, and Model's (Case

VII.) cases. In Caussaude's case there is onset after the third attack, with signs of massive pneumonia. In Roque's case influenza seemed to be etiological.

Chemical Irritants. In Schmidt's case the onset was after inhalation of smoke at a fire.

Family History. There is a family history of tuberculosis in Schittenhelm's and Beshorner's cases.

Personal History. An almost invariable past history is chronic bronchitis; duration varies much in different cases. In Brannan's case there was a history of angioneurotic œdema, and the author thinks that possibly the condition is the result of angio-œdema of the bronchi. In Mader's case there was a history of pemphigus previous to the onset of the attack, which condition extended to the mucous membranes of the mouth, throat, and larynx. The author is inclined to think that all cases represent a pemphigus of the bronchi.

Symptomatology. The onset is usually with an exacerbation of a chronic catarrh; some fever may accompany it. In Caussaude's case the onset was with a massive pneumonia at the left base. In Roque's case there was a long period which preceded the fibrinous expectoration, during which the patient had cough and night-sweats, and gradually became emaciated. A diagnosis of neurasthenia following influenza was made.

Duration—Interval Between Attacks and Duration of Attacks. The one most common characteristic in all the cases outside of the fibrinous expectoration is its tendency to recur in distinct attacks separated by intervals of freedom of longer or shorter periods. It is difficult to give the average duration of the disease, for, of course, many of the cases are lost sight of. Kisch's case is the longest in the history of the disease, lasting over a period of twenty-five years.

Dyspnœa. Paroxysmal attacks of dyspnœa generally immediately precede the fibrinous expectoration; but, strange to say, there are several cases in which the author distinctly states that there was very little dyspnœa: Singer, Model (Case VII.), Schmidt, Pousselt and Zenker (two cases). Evidently the respiratory distress is due entirely to bronchial obstruction. Between the attacks in which the larger complete branching casts are expectorated smaller ones or incomplete portions of casts are brought up, causing, as a rule, very slight dyspnœa. In some cases the expectoration of fragments of casts occurs over long periods before the complete casts appear: thus, in Regardo's case for a period of six years smaller non-branching cylindrical structures compared to macaroni were expectorated, causing considerable dyspnœa.

Cough. Paroxysms of coughing accompany the paroxysms of dyspnœa, and are proportionally intense, but in those cases in which the expectoration of casts is not associated with dyspnœa there may be very little or no cough. Several authors mention that the simple "rauspern" was all that was necessary to cause the expectoration of casts.

Fever. In six cases the paroxysmal attacks of coughing and dyspnœa were associated with slight fever—in Zenker's (Cases I. and II.), Hall's Bernoulli's, Caussaude's, and Street's cases. The fever range was high. Fever may be said to rarely accompany the attacks. Possibly this might point to a non-inflammatory etiology of the disease in its chronic idiopathic form.

Hæmoptysis. In only two cases was hæmoptysis associated with the expectoration of casts—in Sokolowski's first case and Street's case. In the latter there is suspicion of pulmonary tuberculosis from the history, and only one hæmoptysis took place while under observation—a period of six months. In Sokolowski's case, however, the hæmoptysis was copious and usually accompanied the expectoration of casts, which took place daily for a period of nine months. In Schittenhelm's case the hæmoptysis began at the onset of a florid tuberculosis which complicated the fibrinous bronchitis, resulting fatally. Stirling's case on two occasions had hæmoptysis amounting to one ounce, but the author does not say whether there was any association with expectoration of the casts.

In Model's (Case VII.), Mader's, Regarde's, and Worthington's cases the casts were occasionally blood-tinged. Hæmoptysis may be said, then, to rarely accompany the expectoration of casts.

Physical Signs. It is impossible to make a diagnosis from the physical signs. Theoretically if the casts were *in situ* in the bronchi—that is, before the expectoration—the physical signs would be those of what the Germans call "bronchial stenose"—that is, impairment or absence of breath and voice sounds in an area of the lung that is obstructed, with no change in the percussion note. Impairment of resonance is often noted, however, in an area apparently involved, and is usually explained on the ground of collapse of the lung as a result of persistent bronchial obstruction. All types of râles are heard, from crepitant to the large, dry, sibilant râles. The most characteristic adventitious sound is the so-called "ventilgeraüsch" of the Germans, "bruit de drapeau" of the French. This is a very coarse, dry, clicking sound, apparently from its character, caused by the flapping to and fro of loosened portions of the cast with inspiration and expiration. Model, Beshorner, and Caussaude have noted it especially in their case before the expectoration of the casts. Localization of the sign is usually in the lower portions of one or both lungs. Chronic emphysema with its characteristic signs is often associated.

General Symptoms. In several cases a considerable degree of loss of weight and strength has been noted (Baumgarten, Championnière, Street, Waldenburg, Sokolowski, and Roque). Baumgarten attributes the emaciation in his case to the withdrawal of alcohol. Sokolowski's is attributable to the frequent hemorrhages from the lungs. Undoubtedly, however, the emaciation is not an uncommon symptom, due in most cases to the very frequent and exhausting attacks of coughing and dyspnœa. Night-sweats without any discoverable tuberculosis have been associated in a few cases. (Roque and Street.)

Subjective Symptoms. A feeling of oppression and tightness in the chest is the rule, with complete relief on expectoration of casts. The patient can often tell from which portion of the chest the casts come by a peculiar subjective sensation in one region. (Schmidt and Caussaude.) Pain is not a common symptom, though it is marked in some cases.

Complications, Sequelæ, and Results. Complication with some form or other of skin disease have been noted frequently. Thus in Mader's case pemphigus of the skin and mucous membranes preceded the attack of fibrinous bronchitis. In Waldenburg's case an impetigo capitis was noted six months after the symptoms had disappeared. Street's case was complicated by herpes zoster over the shoulder, and later by an eruption of an impetiginous character over the forehead. During the prevalence of the eruption the patient was free from attacks of fibrinous bronchitis.

In three cases the patient was under observation at the time of death. Schittenhelm's case died as the result of florid tuberculosis. The occurrence of fibrinous bronchitis in the course of tuberculosis is common, as will be seen later. This is the only case, however, of tuberculosis supervening on an attack of fibrinous bronchitis. Sokolowski's case was not autopsied. He died as a result of emaciation and anæmia. Mader's case died six months after the last attack of fibrinous bronchitis, the cause being laryngeal stenosis due to cicatrization of a laryngeal ulcer and hypostatic pneumonia. In only one case (Sokolowski's) was a fatal termination the direct result of the disease.

Macroscopical Characters of Casts. In Schittenhelm's case the casts were 10 to 15 cm. long. In Pousselt's case the picture of a cast 16 cm. long is given; in Beshorner's case, 10 cm. long; Stirling's case, 3 to 4 to 6 inches long; Kisch's case, 3 to 8 cm. long; Bernouilli's case, one cast 10 cm. long with ten branches. Championnière gives an excellent picture of a cast 10 cm. long; Waldenburg's case 4 to 5 inches long. All authors who give accurate descriptions note the lumen sometimes in the larger, sometimes in the smaller branches. Championnière noted the separate whirls inside of an outer laminated layer. Dr. Osler examined the casts in Worthington's case and states that "each cast consists of a variable number of laminæ arranged concentrically, those in the centre being much folded and involuted." Most of the branches are cylindrical, some of the larger ones are flat.

Irregularities on the surface of the cast are often noted, and are supposed to be due to bubbles of contained air.

Microscopical Characters of Casts. In eight out of the twenty-seven cases no detail is given as to the microscopical structure of the casts. The usual description is of a fibrillar stratified ground substance containing in its meshes leucocytes. Pousselt, Schittenhelm, and Model are the only authors who state definitely what the characters of these leucocytes are, and they found them to be for the most part mononuclear leucocytes. Sokolowski, Kisch, and Stirling found red corpuscles. Schmidt finds peculiar bodies in the casts which he considers analogous to the rows of red blood-corpuscles, such as are seen in thrombosed capillaries. He also finds corpora lecithinoidea. Alveolar epithelial cells are commonly found, but no authors mention cylindrical epithelial cells, and in my case, after a careful search through many sections, I did not find one. No authors reporting cases in this group mention eosinophiles. In Model's (Case VII.) and Waldenburg's case there were very numerous fat-droplets throughout the casts, and in Model's case the milky appearance of the sputum led the author to suspect that his patient was fooling him. His conclusions with reference to his case are that it represents a lymphorrhagia or chylorrhagia, for (1) a chemical analysis of the sputum and casts gives the same percentage of fat as is found in chylous exudates; (2) the cells in the casts were mostly lymphocytes; (3) there was no dyspnœa or fever associated with the expectoration; (4) the remissions over long periods are exactly similar to those seen in lymphorrhagia or chyluria. He concludes generally that many cases of fibrinous bronchitis may represent a chylorrhagia or lymphorrhagia, for (1) it is in many cases secondary to disturbances of circulation; (2) the absence of epithelium in the casts and the intact mucous membrane found at autopsy point to this explanation.

Charcot-Leyden crystals were found in both of Zenker's cases. Five authors state definitely that they found none. Other authors do not mention these structures. We shall hear more of this in connection with the cases related to asthma.

Chemical Composition of Casts. Most authors simply state that the main mass of the casts were composed of laminæ of fibrin, being influenced wholly by the past views with reference to this and the laminated structure and general consistency of the casts. Championnière (1876) was the first to suggest that the casts were not composed of fibrin but of mucin. He says that microscopically it has not the reticulated appearance of fibrin "mais bien l'aspect uniforme et presque hyalin des matieres muqueses." It resists the action of acetic acid. He noted little irregular whirls enclosed by an outer lamina, which whirls he considered to be mucous moulds of the bronchial glands. Caussaude (1889) came to the conclusion that the casts were composed of syntonin from the following data:

a. Are insoluble in lime-water; do not swell in water at a temperature of 45°; are soluble in acetic acid; are therefore not mucin.

b. Insoluble in sulphate of sodium and chloride of sodium and its deposit in very irregular whirls and circles, and the fact that it does not take the Weigert stain. It is, therefore, not fibrin.

c. Put in the incubator and immersed in a 1 : 1000 solution of hydrochloric acid at the end of twenty-four hours some solution had taken place.

The filtrate when neutralized gave a precipitate which redissolved on the addition of lime-water. Are, therefore, syntonin.

Stirling stated that they consisted of coagulated albumin, soluble in alkalies. Beshorner (1893) states that the casts are not fibrin, for lime-water dissolves them; they do not swell up in acetic acid, and, excepting a few fibrils, they do not take the fibrin stain. The fibrillæ that do take the stain look like the fibrillæ in the central thread of Curschmann's spirals. The author concludes that the exudate is like that in enteritis pseudomembranacea. Pousselt describes portions that take the fibrin stain enclosing mucus which does not take the stain, and is surprised that sections at different levels show portions morphologically similar, some of which take the fibrin stain and some of which do not. Schittenhelm concludes that chemically they are fibrin, for they digest in artificial gastric juice and decompose in hydrogen dioxide. The fibrin stain shows portions which take the stain and which enclose mucus in which are embedded cells.

Study of Bronchi at Autopsy. Schittenhelm's case was fatal as a result of florid tuberculosis. He could determine no especial localization of the tubercles about the bronchi. He found the alveoli and the bronchi of the right lower lobe filled with desquamated epithelial cells and fibrin. The bronchial mucous membrane showed nothing except dilated mucous glands which contained desquamated cells and mucus. The peribronchial tissue showed dilated capillaries infiltrated with leucocytes. Some of the bronchi of the right lower lobes contained plugs, but no definite branching casts. The fibrin stain gives fibrin fibrils throughout the tissue of the lungs, especially about the tuberculous area (but the author does not lay stress on this, as it is found in uncomplicated tuberculosis). Streptococci are seen free in the lumen and in the glands of the bronchi, but none are found in the tissues. No pneumococci, no diphtheria bacilli. He concludes that the disease is a result of a desquamative catarrh of the alveoli of the lobe of the lung, and an acute exudation into the bronchi, bronchioli, and alveoli.

GROUP II. The second group of cases, the acute cases with expectoration of branching casts, number fifteen.

Etiology. Sex: Males, 11; females, 3; (?) 1.

Age: First decade, three; second decade, six; third decade, one; fourth decade, four; seventh decade, one. There is no striking preponderance in youth.

Occupation: Occupation was deemed etiological in the following cases: Glasgow's case, a painter; Ott's case, a knife-sharpener; Sokolowski's case, a physician who was doing bacteriological work, and the author thinks it was a laboratory infection, as he cultivated streptococci and staphylococci from the sputum.

Acute Diseases. (*a*) *Pneumonia.* Koch's case, a man, aged eighteen years, had suffered three attacks of pneumonia, the last of which was characterized by great dyspnœa, a great deal of cough and expectoration, the latter containing numerous non-branching "gerinnsel." Temperature fell on the seventh day by lysis, and six days later the patient was seized with a violent pain and a paroxysm of intense dyspnœa and coughing, which was relieved by the expectoration of three or four branching casts. In Ott's case the expectoration of branching casts preceded the pneumonia; there was no expectoration of casts during the disease. He cultivated the staphylococcus pyogenes aureus and Fränkel's pneumococcus from the casts.

(*b*) *Typhoid Fever.* In Moller's case there was expectoration of casts coming on twenty-three days after the onset of so-called "typhoid." The diagnosis was perhaps doubtful.

In Souque's case, on the thirtieth day of a typhoid complicated by cutaneous abscesses which kept the temperature slightly elevated, there was a paroxysm of intense dyspnœa which was sufficient in duration to make tracheotomy necessary. No relief was experienced from this operation, and the tube was removed. From the tracheal wound a branching cast 15 cm. long was coughed up. There is no further history.

In this connection I might mention a case which I have not classed in any group, but which is both interesting and unique. This is the one reported by Eisenlohr. A typical case of typhoid with considerable bronchitis at the onset and expectoration, without respiratory distress or effort, of exquisitely branching casts on the second, third, fourth, and sixth days after the onset. The casts dissolved in limewater and contained red blood-corpuscles, leucocytes, and alveolar and cylindrical epithelium.

(*c*) *Scarlet Fever.* (Adserson.) Scarlet fever followed by bronchopneumonia preceded the fibrinous bronchitis by some months.

(*d*) *Measles.* (Yäger.) Onset two days after convalescence from measles.

Basedow's Disease. Fritsche reports a case in the course of Basedow's disease.

Symptomatology of the condition as concluded from twelve out of the fifteen cases is somewhat as follows. (We exclude Koch's case following pneumonia; Souques', in typhoid, and Vintras' case.)

Onset. Preliminary acute bronchitis in eight cases varying in duration; in Herzog's case it had existed four weeks before the expectoration of casts. The characteristic expectoration usually appears about the end of the first week.

Chills. In Bettelheim's case chill at onset, followed three days later by the expectoration of casts. In Kretschy's case the patient had a chill every evening up to the fifth day of the attack. He died on the eleventh day. The author thinks it represents an acute infection.

Glasgow's case: A chill just before the expectoration of casts.

Fever. The fever range is high, in Kretschy's case averaging 40° C. in the evening. This high fever preceded the expectoration of casts by five days. In Fritsche's case the temperature was 40° C. in the first attack.

Distinct Attacks. The disease shows the same periodicity that is exhibited in the chronic form, consisting of a number of distinct attacks characterized by fever, dyspnœa, cough, and expectoration of casts. In the interval between the attacks the patient is comparatively comfortable, though still febrile. In Bettelheim's and Herzog's cases there was only one such attack, but in the latter case it was preceded by a fever and catarrhal symptoms in the bronchi, lasting four weeks. In Kretschy's case there were only two attacks, occurring on the fifth and seventh days of the disease. In Glasgow's case there were two attacks with a long interval between the attacks, the second attack being marked by freedom from dyspnœa and great ease in expectoration. It is quite common in all cases to have fragments of casts expectorated for long periods before and after the characteristic expectoration. In Fritsche's case there were two attacks with an interval of four days. The paroxysms generally occur in the morning.

Pain. The pain in the thorax is usually very intense during attacks. A feeling of oppression in the chest exists between the attacks.

Hæmoptysis was found in only two cases—Bettelheim's and Herzog's.

Number and Macroscopical Structure of Casts. The casts are remarkably few in number. In Bettelheim's case only two complete casts were expectorated. In Kretschy's case only four (duration, eleven days, resulting fatally). Macroscopically there is no difference from those seen in the chronic form.

Microscopical structure does not differ from those seen in the chronic form. Koch found Charcot-Leyden crystals in the casts. Adserson, Ott, and Vintras state definitely that these structures were not present. Herzog, from staining reactions and chemical tests, concluded that the casts were fibrin. Koch found that most of the cells were mononuclear leucocytes. In Vintras' case the casts dissolved in lime-water. Souques thinks they were fibrin from their morphological appearance, but states that they did not give the fibrin reaction.

Result. Naturally some of the cases in this group might very well be the first attack of a condition which was to become chronic. Bettelheim's and Glasgow's cases suggest this, for after the subsidence of the acute attacks they had slight attacks for a month or so. In several of the chronic cases the first attack is very like in symptomatology to the acute form, and undoubtedly there are transitions between the two which might well be classed as subacute.

In the fifteen cases here considered four were fatal and three autopsied.

Autopsies. 1. Kretschy's case. Young man. Cast found *in situ* in right middle and upper lobes. The mucous membrane of the bronchi of one lung showed loss of epithelium and infiltration with white cells. The other lung showed œdema.

2. Yäger's case. Child, aged five years, died, the expectoration of casts diminishing with increasing dyspnœa and aphonia. Section showed a fresh adhesive pleuritis and pericarditis--bronchopneumonia of the left lung—a weak, incompletely adherent deposit in the larynx, which became thicker and more solid as bifurcation was approached, and extended into all the branches of the bronchi of the left lung and a little way into the right. The case followed an attack of measles. In its autopsy finding and clinical course it is almost identical with Hilton Fagge's case, which we have quoted above.

3. Vintras' case is unique in the history of this disease. An emaciated soldier, aged sixty-eight years, had suffered from cough for three months before admission. He had copious hæmoptysis a week before admission. Four days before admission an attack of intense cyanosis and dyspnœa relieved by expectoration of a branching cast. Seven days later another attack, and fifteen casts were coughed up. Seven days later the patient died in another attack.

At autopsy a rapidly growing epithelioma was found in the œsophagus opposite the bifurcation of the trachea, which latter would hardly admit a goose-quill. A cast was found projecting from the right bronchus into the trachea and obstructing the already strictured lumen of the latter. One small cast was also detected in the tubes of the right middle lobe.

GROUP III. The next group of cases includes those cases in which casts were found at autopsy, but were not expectorated during life. They number six. Clinically they do not belong to the group of fibrinous bronchitis. The probability is that many such cases go undiagnosed because no autopsy is made. Hutchinson's case is probably a case of diphtheria of the larynx in which extension of the membranes into the bronchi followed tracheotomy.

The two cases reported in Virchow's *Archiv* (1879) followed rupture of caseous lymph glands into the bronchi, both in very young children. Escherich's case was a fatal case of double pneumonia. Mazotti's case, a woman, aged seventeen years, died at the end of two weeks with high fever and dyspnœa. At autopsy the lesions of typhoid were found and a fibrinous inflammation of the bronchi. (This makes in all four cases showing an association with typhoid.)

Cutler's case is the only idiopathic case in the group. A woman, aged sixty-five years, had had acute bronchitis for three days; intense dyspnœa and cyanosis supervened, resulting in death in three hours. Section showed " pharynx and larynx healthy. A croupous membrane lay reflected on itself over the entrance of the two primary bronchi. When laid in place this membrane reached more than half-way up the trachea. Downward it extended into the minutest divisions of the bronchi, in many of them forming an almost solid plug. The alveoli contained no solid matter, and except in a few places where there was œdema and collapse they contained air." Evidently in these cases death occurred from asphyxia, because the casts were not expectorated. In this connection the rather bad prognosis of the disease in the two extremes of life might be mentioned, possibly having some relation to the fact that expectoration is difficult at these periods. A study of the cases in this group and the fatal cases of the second group shows the following facts: Out of twenty-one cases ten were fatal.

In the second group, Möller's case, aged thirteen years, not autopsied. Kretschy's case, a boy, autopsied; cast in bronchial tree. Yäger's case (measles), aged five years, autopsied; cast in bronchial tree. Vintras' case (epithelioma), aged sixty-eight years, autopsied; cast in bronchial tree.

In the third group, Cutler's case, aged sixty-five years, autopsied; cast in bronchial tree. Mazotti's case (typhoid), aged eighteen years, not autopsied; associated with typhoid. Escherich's case (pneumonia), aged twenty-seven years, autopsied; casts in bronchi. Virchow's *Archiv* (rupture of bronchial lymph gland), two cases, aged four months and four years respectively, autopsied; casts in bronchi. Hutchinson's case (diphtheria), child, autopsied; casts in bronchi and trachea.

GROUP IV. The fourth group of cases includes all those in which, although no definite arborescent moulds of the bronchial tree were expectorated, fibrinous structures evidently emanating from the bronchi were: eleven cases.

The symptomatology in some of the cases is very much like the symptomatology of the cases in the first and second groups. The difference is, perhaps, only a matter of degree, in this group the exudation being less in amount, perhaps more limited in area, and the expectoration taking place before a definite branching cast is formed.

In going over these cases one is impressed with the fact that the authors in many instances have reported in the same article cases belonging to the first and second groups. Thus have Chvostek, Kretschy, Model, Sokolowski, and Chauffaud. The natural inference is that these authors, having observed one case in which the expectoration contained these wonderful arborescent casts, kept their eyes open for other similar cases. It is well to separate these cases from those in the other groups to bring out the frequency of the cases in which arborescent casts are expectorated, or, rather, the infrequency. It might be mentioned that fibrin is frequently present in the expectoration in ordinary bronchitis. I have a specimen of a plug expectorated in a chronic bronchitis showing quite a marked content in fibrin (Weigert's stain). There are eleven cases in the group.

GROUP V. The fifth group of cases includes all those associated with organic heart disease—ten cases. There is no good reason why the cases in this group should not be included in the two groups of idiopathic cases. Heart lesions predispose to bronchitis through congestion. If this bronchitis assumes a special type there is no reason to suppose that the heart lesion is the etiological factor. We certainly consider the bronchitis of heart disease as an idiopathic bronchitis, though recognizing that a predisposing factor lies in the organic heart lesion. In Bernouilli's, Degen's, Lawrence's, Hint's, and Habel's cases the condition was chronic. It is worthy of note that all these cases were afebrile, and in three of them dyspnœa was absent during the attacks. This might indicate that the process was purely mechanical, and not inflammatory, the exudate taking place quickly and as a result of congestion. Seven of the cases were under observation at the time of death. In Degen's case, in a boy, aged sixteen years, the duration of the disease had been eight years; the autopsy findings were adherent pericarditis externum with exudation, aortic and mitral insufficiency; brown induration of the lungs. The right main bronchus and its branches contained a cast *in situ*, also the bronchi of the left lower lobe. Rohr's case: a woman, aged twenty-one years, under observation during the last month and a half of life, during which time she had loss of compensation and three attacks of fibrinous bronchitis. At autopsy: mitral insufficiency, infarct of lung, no casts, a tubercular kidney.

Beshorner's case; a male, aged forty-nine years. No clinical history. At autopsy a pleuritic exudation, adhesive pericarditis, nephritis, and peribronchitic infiltration of the lungs. It is worthy of note that in three out of the five cases that were autopsied adherent pericarditis was found. In Habel's, Hint's, and Schittenhelm's cases (all of which were under observation at the time of death), when compensation was lost, there were no longer any attacks of fibrinous bronchitis. Schittenhelm's case resembled an acute fibrinous bronchitis, the other two were chronic.

Structure of Casts. The four authors who investigated carefully found that the casts were not in the main composed of fibrin. Grandy and Beshorner found that the casts did not take the fibrin stain at all. Habel found a few fibrillæ staining for fibrin. Hint concludes that it was mucin not fibrin on rather insufficient data. No authors mention Charcot-Leyden crystals or eosinophiles.

Study of Bronchi at Autopsy. Grandy made a very careful study of the condition of the bronchi and of a cast *in situ.* He found dilatation of the vessels of the bronchi, clumps of leucocytes especially about the bases of the mucous glands, which latter were much dilated and filled with mucus, and it could be seen that this mucus merged into the casts. There were very great numbers of goblet cells present. The character of the staining reaction of the mucus was the same as that of the outer layer of the cast (blue with Van Gieson, red with thionin); but in the inner part the reaction had changed somewhat, showing that the mucus had altered in character.

Heart Conditions in this Group. Mitral insufficiency in five cases; mitral insufficiency and stenosis in one case; mitral stenosis in one case; mitral insufficiency and adherent pericardium in one case; adherent pericarditis in two cases.

GROUP VI. The sixth group includes those cases associated with tuberculosis of the lungs. There are fourteen in all, ten of which were under observation at the time of death, and were autopsied.

The expectoration of casts generally occurs late in the disease. In Homolle's case two casts were expectorated, one eight months before death and one eleven days before death. In Fraentzel's case casts were expectorated for the last seventeen days of life. In Pramberger's case a cast was expectorated fifty-five days before death and one eleven days before death. Jaccoud's case just one day before death expectorated two casts. Model's case (Case IV.) expectorated casts for two months before death. Model's case (Case V.) for ten days previous to death. Sokolowski's case, thirty days before death expectorated one cast. Casts were found at autopsy *in situ* in three cases—Homolle's, Model's (Case V.), and Klein's cases.

Model found a caseous infiltration of the walls of the right upper bronchus in which a cast was found *in situ.* Magniaux found peribronchial miliary tubercles in both apices, with denudation of epithelium and infiltration with leucocytes.

Structure of Casts. The casts are very few in number, and are the typical large branching structures found in the idiopathic form. All authors in this group state on rather insufficient evidence that the casts are composed of fibrin. Klein is the only author who goes carefully into the composition, and he finds that it does not take the fibrin stain and that it dissolves in chloroform. No authors mention staining sections of the casts for tubercle acilli, so that if these peculiar bodies which we have described in our cases were present they were overlooked. Tubercle bacilli were always found in the sputum.

All possible tuberculous conditions of the lung are found in this group of cases. Magniaux cultivated the diplobacillus of Friedländer from the blood, organs, and casts.

GROUP VII. Occasionally in the literature on bronchitis fibrinosa there is a reference to the relation of this disease to asthma. The suggestion of a relationship is brought about by the fact that there is a group of cases in which small fibrinous structures are expectorated along with the common contents of asthmatic sputum, Charcot-Leyden crystals, Curschmann's spirals, and eosinophiles. Are these cases to be classed as asthma or as bronchitis fibrinosa?

Schmidt has made elaborate studies into the sputum in asthma, and he found more or less large fibrinous structures in six out of eight cases of asthma which he investigated, using Weigert's stain to demonstrate the fibrin. He gives two cases especially in which the "gerinnsel" were quite large—2 to 3 cm. long. He describes them as "fädige und flocksige gerinnsel," which were composed for the most part of fibrin, and also contained some mucus in spiral arrangement, many eosinophilic cells, and numerous crystals. One of these cases was associated with hypertrophic rhinitis, and suffered typical asthmatic attacks. His conclusion is: "Berucksichtigt man aber, das wiederholt auch bei fibrinöser Bronchitis Spiralen gefunden worden sind, so durfte es in der that schwer sein, Fälle wie diesen, welcher dem Sputum nach eher zur fibrinöser Bronchitis, dem klinichen Bilde nach zum Asthma gerechnet werden muss, zu classificieren." The author also found small fibrinous structures in two cases of ordinary bronchitis. He concludes that the exudation of fibrin is quite common in inflammatory conditions of the bronchioles. (My preparation under the microscope is a plug from a case of ordinary bronchitis in emphysema. It shows fibrin in fair amount. Demonstrated at the Johns Hopkins Medical Society, June 2d.) Posselt makes an elaborate analysis of many cases of bronchitis fibrinosa from the point of view of the relation of this disease to asthma. He concludes that the cases reported as fibrinous bronchitis can be classified into two distinct groups:

(*a*) "One in which the casts consist almost exclusively of fibrin containing very few cells and little or no mucus, and in which the expectoration takes place without any asthmatic attacks or the least subjective inconvenience. A relationship to any other pathological condition is not demonstrable. The etiology is obscure. There are very few cases of this essential primary fibrinous bronchitis. The course is chronic, with subacute, rarely acute attacks, in which casts are expectorated."

(*b*) "Second group. Very much more numerous than the cases of the first group. There are cases in which the fibrinous bronchitis is complicated by an asthma in which the casts are rich in cells and crystals, and spirals are often found. The casts are also rich in mucus. The course is much more severe and acute." I will not tire you with the gross misstatements and contortions of fact upon which this classification of Pousselt's is based. They are numerous, occupying about one hundred pages. His other conclusions are that bronchitis fibrinosa is often complicated by asthma, but that the two processes are entirely distinct—the former not being in any case an extension of the asthmatic exudation into the larger bronchi. However this may be, the fact remains that there is a well-marked group of cases in which the so-called "asthma-cueger," Charcot-Leyden crystals, Curschmann's spirals, and eosinophilic cells are expectorated along with small fibrinous structures representing small casts of the finer bronchi, but generally not branching. The amount of dyspnœa in the attacks in these cases is much greater than the size and number and form of the casts would indicate if one looked to *bronchial* obstruction as the etiological factor in causing the dyspnœa. One is, therefore, tempted to classify these cases as asthma in which the exudative process has extended beyond the bronchioles in some areas of the lung, causing the formation of casts, generally not branching in some of the smaller bronchi. "Leyden has stated that one might consider asthma as a fibrinous bronchiolitis which occasionally can extend to the large bronchi, so that large 'gerinnsel' are expectorated. Nevertheless, one cannot identify asthma with fibrinous bronchitis, for there are many cases of the latter affection which have nothing to do with asthma. The cases which are related to asthma are characterized by the fact that the casts are filled with Charcot-Leyden crystals. Fränkel agrees with Leyden as to the relationship of the two diseases, but does not think asthma should be designated a fibrinous bronchiolitis, for the exudate is not for the most part fibrin." (From Pousselt.)

In going over the cases since 1869 I find that in five out of the seven cases in which Charcot-Leyden crystals have been found in the casts the latter have been small, generally possessing very few or no branchings. I am, therefore, tempted to place these in a group by themselves, showing a transition between fibrinous bronchitis and fibrinous bronchiolitis (asthma). They certainly represent a group distinct from the first and second groups, in which the casts are very large and show exquisite and numerous dichotomous branchings.

In Vierordt's case Curschmann's spirals appeared after the fibrinous structures had disappeared, thus suggesting the alternate occurrence of asthma and fibrinous bronchitis.

I am fortunate in being able to present, through the kindness of Dr. Osler, a case which I think represents this group well, and will bring in marked contrast the case spoken of earlier in the paper.

Kate M., aged forty-five years, white; first admission to hospital March 6 to 15, 1900. Complaint: Cough for several years, and lately some smothering sensation on lying down.

Family History. Father died of "asthma." Mother died of dropsy. Two sisters died of consumption.

Personal History. Has had eight children. Had a severe attack of rheumatism eight years ago. Is frequently a sufferer from tonsillitis. During the last sixteen years Dr. Mackenzie has removed about twenty polyps from the nose.

Present Illness. Has suffered from cough for periods throughout the last twenty years. The cough comes on in paroxysms at night, and is associated with attacks of shortness of breath. She says she can bring on a paroxysm by spraying her nose. She brings up considerable expectoration during these attacks.

Physical Examination shows typical signs of mitral insufficiency, with good compensation. Herpes about the lips and nose; tonsillitis while in the hospital; no note on the sputum; trace of albumin in the urine; hæmoglobin, 68.7 per cent; no temperature.

Second admission, April 15th; June 2, 1901. Complains that she has never been free from cough since she left the hospital. For the last two months this cough and "smothering feeling," as she calls it, have been worse.

Present Illness. Has been kept in bed on account of her heart lesion ever since entrance to the hospital. During this time she has had frequent attacks of dyspnœa and coughing and considerable amount of expectoration. No fever has been present. The attacks usually occur in the early morning. The respiratory distress is extreme, and vomiting is often associated with the expectoration. In the interval between these paroxysmal attacks she has, as a rule, been free from dyspnœa, though at times she has suffered considerably from it, having to remain in a reclining position in bed.

Physical Examination shows the signs of a chronic bronchitis; a well-marked mitral insufficiency; no signs of loss of compensation; hæmoglobin, 47 per cent.; no albumin in urine.

Sputum. Sputum contains Curschmann's spirals in abundance, eosinophilic cells, no tubercle bacilli. There are a fair number of very small branching casts, none over 3 cm. long, the majority averaging not over 1 cm. in length and only showing one or two incomplete branchings. Curschmann's spirals are seen at the end of the finer branches very frequently, and suggest the relationship spoken of above between bronchiolitis fibrinosa (asthma) and bronchitis fibrinosa. The calibre of the largest cast is not over 2 mm., and no lumen is seen on cross section. The casts consist for the most part of fibrin (as shown by Weigert's fibrin stain), containing leucocytes, generally polynuclear eosinophiles. No Charcot-Leyden crystals are seen. Sections cut longitudinally through the branches which end in Curschmann's spirals show that the fibrin from the casts is continuous with the fibrin in the central thread of the spiral. Streptococci and staphylococci are numerous in the casts.

GROUP VIII. The eighth group of cases includes those associated with acute œdema of the lung after thoracentesis. There are four such described by Ortner. In two of the cases fibrinous casts were expectorated (Hampeln and Ortner's second case), associated with a great amount of albuminous expectoration. In Ortner's case only one cast 3 cm. long was expectorated. The patient, a woman, died two months later with increasing loss of compensation; adherent tuberculous pericarditis, double tuberculous pleurisy, œdema of the lungs, congestion of the liver, and ascites were found. At autopsy there were no casts in the bronchi. Ortner and Scriba describe cases which resulted fatally in which casts were not expectorated, but were found *in situ* at autopsy. In Ortner's case, in the right lung; in Scriba's, in the left, corresponding to the side on which thoracentesis was performed. In Ortner's case the autopsy findings were adherent pericarditis, mediastinitis, congestion of the liver, and ascites. In Scriba's case tuberculous pleurisy and pericarditis were found at autopsy. In this connection it is interesting to recall that in three out of the five cases of fibrinous bronchitis associated with heart disease which were autopsied, adherent pericarditis was found; also in one case in the second group adherent pericarditis was found.

GROUP IX. The ninth group includes a few cases which I have been unable to classify through dearth of description. (See References.)

In going over the literature I came across an interesting and unique case reported under the title of "Bronchite Pseudomembraneuse Aspergillaire Primitive," by Devillers et Renon, in *La Presse Médicale* for 1899. It affected a woman, aged forty years, who coughed up for a considerable time greenish membranes emanating from the bronchi, entirely composed of mycelium of aspergillus fumigatus. Her profession consisted in selecting seeds for cultivation. Her method of testing them was to feel their consistency with her teeth. It is supposed that she thus acquired this peculiar infection. Attacks of dyspnœa and cyanosis preceded the expectoration.

Expectoration of blood-casts of the bronchi following hæmoptysis is rather rare, and in consulting the literature I came across only three cases the references of which are given.

NOTE I.—*Note with Reference to the Relation of Pregnancy and Menstruation to Fibrinous Bronchitis.*

No such relation was made out in the case of Maggie S. Schnittzler (1864) observed a case. A woman after her sixth labor, instead of having her menstrual flow at the eighth week (as was her custom), had an attack of fibrinous bronchitis. For several months this phenomenon recurred. After her seventh and eighth labor the author made the same observation. This same case was again observed by Brik seventeen years later, when the woman was fifty-nine years old, in an attack of fibrinous bronchitis. Evidently at this age neither pregnancy nor menstruation could have had an etiological influence.

Posselt quotes Stofella, who observed a case in which attacks of fibrinous bronchitis replaced menstruation.

NOTE II.—*The Bacteriology of Fibrinous Bronchitis.*

In the cases considered in this paper the following bacteriological facts have been elicited:

GROUP II. Souques demonstrated streptococci, pneumococci, and staphylococci in the casts. Ott cultivated the staphylococcus aureus and the pneumococcus from the casts. Herzog demonstrated staphylococci and bacilli in the casts. Koch found bacteria which retained the stain by Gram's method always enclosed in mucus. He therefore does not consider them etiological.

GROUP IV. Sokolowski cultivated the staphylococcus albus and aureus from the sputum—a fact having no bearing whatsoever.

GROUP IX. Sokolowski demonstrated staphylococcus and streptococcus and a tetragenous conus in the casts. Neither diphtheria bacilli nor tubercle bacilli have ever been demonstrated in the casts.

Landireux and Triboulet (reference given under "Foreign References not Read") cultivated pneumococcus from the casts. It is an accepted fact that staphylococcus, streptococcus, and pneumococcus are etiological in the production of simple purulent bronchitis, so that their presence in the casts does not wholly explain the etiology of this rare disease. One observer has gone so far as to claim that in a case of his antistreptococcic serum was curative. Hoffmann (Nothnagel's *Handbuch*, xiii. Bd., iii. Th., 1 Abth.) recognizes two organisms as etiological: the pneumococcus and the diphtheria bacillus. He admits that the pneumococcus can be demonstrated in simple purulent bronchitis, but ventures the conjecture that cases in which it is alone or principally active develop fibrinous bronchitis; whereas, the purulent form appears where streptococci or other cocci overgrow it. This is only a conjecture.

Pinhini isolated a bacillus in three cases which, inoculated into the trachea of rabbits, practically reproduced the disease. Inoculation into other parts of the body was with negative result. His three cases occurred in three bricklayers who were working in the neighborhood of a sewer, and are supposed thus to have acquired the infection. I have not read the original article, "Contributio allo studio della Bronchite fibrin, acuta prim," in the *Riv. clin. arch. ital. di clin. med.*, 1889, p. 105.

COLLECTIONS OF CASES.

For all references previous to 1869, see Lebert's article. References follow the text, being arranged in groups.

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1873. Bettelheim. Casuistische Mitth. über B. Fibrinosa, Wien. (Schmidt's Jahrbücher, vol. clxiii.)
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1893. Fritsche. Read in Schmidt's Jahrbücher, Bd. ccxxxvii. S. 219.
1897. Allen. Transactions of Medical Society of District of Columbia.
1898. Patton and Herzog. Journal of American Medical Association, vol. xxx. p. 25.
1900. Ott. Deutsch. med. Wochenschrift, xlvii.
1900. Vintras. Lancet, Sept. 15, p. 809.
1900. Souques. La Independence Médicale, Jan. 17.
Case in the course of typhoid fever.
1876. Eisenlohr. Berliner klin. Wochenschrift, No. xxxi.

GROUP III. 6 Cases. *Cases in which casts were found in bronchi at autopsy, but were not expectorated.*

1874. Hutchinson. Trans. of Path. Soc. of Phila. (Probably diphtheritic.)
1879. (?) Virchow's Arch. f. Path. Anat., Bd. lxxii., S. 294 (2 cases).
1883. Escherich. Deutsch. med. Wochenschrift, No. viii.
1884. Mazotti. Bolle dello Scienze Med. di. Bologna, June 14, 1884. Read in Centralblatt f. klin. Med. for 1885.
1887. Cutler. Boston Medical and Surgical Journal, p. 443.

GROUP IV. 11 Cases. *Cases in which the casts expectorated showed no dichotomous branching.*

1875. Chvostek. Wiener med. *Presse.*
1892. Kingsley. St. Louis Courier of Medicine, March.
1882 Madigan. Chicago Medical Review, p. 184.
1883. Kretschy. Deutsch. med. Wochenschrift, No. viii.
1881. Barron. The Lancet.
1889. Gibson. Practitioner, vol. xliii.
1890. Model. "Ueber Bronchitis Fibrinosa." Dissertation, Freiburg.
1893-94. (?) The Hospital, London, p. 240.
1896. Sokolowski. Deutsch. Arch. f. klin. Med., p. 482.
1898. Lepine. Rev. de Méd., 1898, vol. xviii. p. 835.
1899. Chauffarde. Rev. Internationale de Méd. et de Chir.

GROUP V. 10 Cases. *Expectoration of Branching Casts in the Course of Organic Heart Disease.*

1877. Bernoulli. Deutsch. Arch. f. klin. Med., Bd. xx.
1878. Degen. Würtemberger med. Correspondenz-Blatt, Bd. xlviii.
1886. Stack. Berliner klin. Wochenschrift, S. 221.
1892. Rohr. Correspondenz-Blatt f. Schweizer Aerzte, vol. xxii. p. 767.
1893. Lawrence. Lancet, vol. i. p. 247.

1893. Beshorner. Volkmann's Sammlung f. inn. Med., No. iii.
1897. Grandy. Centralblatt f. Allg. Path. u. Path. Anat., vol. viii. p. 513.
1898. Habel. Centralblatt f. innere Med., 1898, No. 1.
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1900. Schittenhelm. Deutsch. Arch. f. klin. Med., p. 338.

GROUP VI. 14 Cases. *Expectoration of Branching Casts in the Course of Pulmonary Tuberculosis.*

1870. Tuckwell. Trans. of Path. Soc. of London (doubtful).
1874. Flint, Austin. Med. Record, vol. ix. p. 41.
1875. Homolle. Bul. de la Soc. Anat., t. x. p. 542.
1878. Fräntzel. Charité Annalen, v. Jahrgang, p. 295.
1881. Pramberger. Mittheil. d. Verein d. Aerzte in Steiermark, 1880, Graz, 1881, vol. xvii, pp. 3-68.
1881. Müller. Hosp. Tidende, vol. ii. p. 12. (Read in Schmidt's Jahrb., vol. cciv.)
1885. Jaccoud. Quoted from Letellier's Thesis, *q. v.*
1890. Model. Ueber Bronchitis Fibrinosa. Dissertation, Freiburg (4 cases).
1895. Magniaux. Thèse de Paris.
1896. Klein. Wiener klin. Wochenschrift.
1895. Sokolowski. Deutsch. Arch. f. klin. Med., lvi. p. 476.

GROUP VII. 5 Cases. *Cases showing a distinct relation to Asthma. Casts small.*

1864. Friedreich. Virchow's Archiv, Bd. xxx. S. 381.
1881. Pramberger. (See Group VI.)
1883. Vierordt. Berliner klin. Wochenschrift, No. 29.
1883. Wolf. Ueber Bronchitis Fibrinosa. Dissertation, Würzburg.

GROUP VIII. 4 Cases. *Fibrinous Exudation into the Bronchi associated with Pulmonary Œdema following Thoracentesis.*

1885. Scriba. Deutsch. Arch. f. klin. Med., Bd. xxxvi. (Cited in Ortner.)
1892. Hampeln. St. Petersburger med. Wochenschrift, S. 336. (Cited in Ortner).
1899. Ortner. Wiener klin. Wochenschrift, No. 41. (2 cases.)

GROUP IX. 6 Cases. *Cases unclassified through dearth of data.*

[illegible] Cohen. Transactions of Path. Soc. of Phila. (Laryngeal Tuberculosis.) Vol. v. p. 126.
1875. Siridez. In Championnière's Thesis, 1876.
1883. Escherich. Deutsch. med. Wochenschrift, No. viii.
1881. Müller. Second Case. Read in Schmidt's Jahrbuch., Bd. 204.
1888-89. Limont. Northumberland and Durham Med. Society.
1900. Lowenthal. Liverpool Medical and Chirurgical Journal.

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1873. Peacock. Transactions of Path Soc. of London, vol. xxiv.
1880. West. British Medical Journal, vol. i. p. 282.
1893. Richards. Lancet, May, 1893.

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1861. Schnitzler. Wiener med. Halle, p. 450.
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